09/687,528

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(FILE 'HOME' ENTERED AT 17:49:38 ON 19 OCT 2004)

FILE 'MEDLINE, CAPLUS, BIOSIS, SCISEARCH' ENTERED AT 17:49:56 ON 19 OCT 2004

L1 216 S RECEPTOR(3A)ADVANCED(W)GLYCATION(W)ENDPRODUCT
L2 85 S SRAGE
L3 283 S L1 OR L2
L4 38783 S RESTENOSIS
L5 9 S L3 AND L4
L6 6 DUP REM L5 (3 DUPLICATES REMOVED)

=> d au ti so pi ab 1-6 l6

L6 ANSWER 1 OF 6 CAPLUS COPYRIGHT 2004 ACS on STN
AU Hudson, Barry I.; Bucciarelli, Loredana G.; Wendt, Thoralf; Sakaguchi, Taichi; Lalla, Evanthia; Qu, Wu; Lu, Yan; Lee, Larisse; Stern, David M.; Naka, Yoshifumi; Ramasamy, Ravichandran; Yan, Shi Du; Yan, Shi Fang; D'Agati, Vivette; Schmidt, Ann Marie
TI Blockade of **receptor** for **advanced glycation endproducts**: a new target for therapeutic intervention in diabetic complications and inflammatory disorders
SO Archives of Biochemistry and Biophysics (2003), 419(1), 80-88
CODEN: ABBIA4; ISSN: 0003-9861
AB A review. The glycation and oxidation of proteins/lipids leads to the generation of a new class of biol. active moieties, the advanced glycation endproducts (AGEs). Recent studies have elucidated that carboxymethyllysine (CML) adducts of proteins/lipids are a highly prevalent AGE in vivo. CML-modified adducts are signal transduction ligands of the receptor for AGE (RAGE), a member of the Ig superfamily. Importantly, CML-modified adducts accumulate in diverse settings. In addition to enhanced formation in settings of high glucose, these adducts form in inflammatory milieu. Studies performed both in vitro and in vivo have suggested that the proinflammatory/tissue destructive consequences of RAGE activation in the diabetic/inflamed environment may be markedly attenuated by blockade of the ligand-RAGE axis. Here, we will summarize the known consequences of RAGE activation in the tissues and highlight novel areas for therapeutic intervention in these disease states.

L6 ANSWER 2 OF 6 CAPLUS COPYRIGHT 2004 ACS on STN
IN Stern, David M.; Schmidt, Ann-Marie; Marso, Steven; Topol, Eric; Lincoff, A. Michael
TI A method for inhibiting new tissue growth in blood vessels in a patient subjected to blood vessel injury
SO PCT Int. Appl., 43 pp.
CODEN: PIXXD2

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 2002030889	A2	20020418	WO 2001-US32036	20011012
WO 2002030889	A3	20020711		

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

AU 2002013192	A5	20020422	AU 2002-13192	20011012

AB This invention provides for a method for inhibiting new tissue growth in blood vessels in a subject, wherein the subject experienced blood vessels injury, which comprises administering to the subject a pharmaceutically

effective amount of an inhibitor of **receptor** for **advanced glycation endproduct** (RAGE) so as to inhibit new tissue growth in the subject's blood vessels. The invention also provides for method for inhibiting neointimal formation in blood vessels in a subject, wherein the subject experienced blood vessel injury, which comprises administering to the subject a pharmaceutically effective amount of an inhibitor of **receptor** for **advanced glycation endproduct** (RAGE) so as to inhibit neointimal formation in the subject's blood vessels. The invention also provides a method for preventing exaggerated **restenosis** in a diabetic subject which comprises administering to the subject a pharmaceutically effective amount of an inhibitor of **receptor** for **advanced glycation endproduct** (RAGE) so as to prevent exaggerated **restenosis** in the subject. In the example provided, a significant reduction in neointimal area was observed in fatty Zucker rats treated with soluble **receptor** for **advanced glycation endproduct** following carotid artery injury.

L6 ANSWER 3 OF 6 MEDLINE on STN

AU Wendt Thoralf; Bucciarelli Loredana; Qu Wu; Lu Yan; Yan Shi Fang; Stern David M; Schmidt Ann Marie

TI **Receptor** for **advanced glycation endproducts** (RAGE) and vascular inflammation: insights into the pathogenesis of macrovascular complications in diabetes.

SO Current atherosclerosis reports, (2002 May) 4 (3) 228-37. Ref: 52
Journal code: 100897685. ISSN: 1523-3804.

AB The incidence and severity of atherosclerosis is increased in patients with diabetes. Indeed, accelerated macrovascular disease in diabetic patients has emerged as a leading cause of morbidity and mortality in the United States and worldwide. Multiple investigations have suggested that there are numerous potential contributory factors that underlie these observations. Our laboratory has focused on the contribution of **receptor** for **advanced glycation endproducts** (RAGE) and its proinflammatory ligands, advanced glycation endproducts (AGEs) and S100/calgranulins in vascular perturbation, manifested as enhanced atherogenesis or accelerated **restenosis** after angioplasty. In rodent models of diabetic complications, blockade of RAGE suppressed vascular hyperpermeability, accelerated atherosclerotic lesion area and complexity in diabetic apolipoprotein E-deficient mice, and prevented exaggerated neointimal formation in hyperglycemic fatty Zucker rats subjected to injury of the carotid artery. In this review, we summarize these findings and provide an overview of distinct mechanisms that contribute to the development of accelerated diabetic macrovascular disease. Insights into therapeutic strategies to prevent or interrupt these processes are presented.

L6 ANSWER 4 OF 6 MEDLINE on STN DUPLICATE 1

AU Degryse B; Bonaldi T; Scaffidi P; Muller S; Resnati M; Sanvito F; Arrigoni G; Bianchi M E

TI The high mobility group (HMG) boxes of the nuclear protein HMG1 induce chemotaxis and cytoskeleton reorganization in rat smooth muscle cells.

SO Journal of cell biology, (2001 Mar 19) 152 (6) 1197-206.
Journal code: 0375356. ISSN: 0021-9525.

AB HMG1 (high mobility group 1) is a ubiquitous and abundant chromatin component. However, HMG1 can be secreted by activated macrophages and monocytes, and can act as a mediator of inflammation and endotoxic lethality. Here we document a role of extracellular HMG1 in cell migration. HMG1 (and its individual DNA-binding domains) stimulated migration of rat smooth muscle cells in chemotaxis, chemokinesis, and wound healing assays. HMG1 induced rapid and transient changes of cell shape, and actin cytoskeleton reorganization leading to an elongated polarized morphology typical of motile cells. These effects were inhibited by antibodies directed against the **receptor** of

advanced glycation endproducts, indicating that the **receptor** of **advanced glycation endproducts** is the receptor mediating the HMG1-dependent migratory responses. Pertussis toxin and the mitogen-activated protein kinase kinase inhibitor PD98059 also blocked HMG1-induced rat smooth muscle cell migration, suggesting that a G(i/o) protein and mitogen-activated protein kinases are required for the HMG1 signaling pathway. We also show that HMG1 can be released by damage or necrosis of a variety of cell types, including endothelial cells. Thus, HMG1 has all the hallmarks of a molecule that can promote atherosclerosis and **restenosis** after vascular damage.

L6 ANSWER 5 OF 6 BIOSIS COPYRIGHT (c) 2004 The Thomson Corporation. on STN
AU Sakaguchi, Taichi [Reprint author]; Sousa, Monica [Reprint author]; Yan, Shi Du [Reprint author]; Yan, Shi-Fang [Reprint author]; Duda, Stephan; Arnold, Bernd; Nawroth, Peter P.; Schmidt, Ann Marie; Stern, David M.; Naka, Yoshifumi
TI **Restenosis**: Central role of RAGE-dependent neointimal expansion.
SO Circulation, (October 23, 2001) Vol. 104, No. 17 Supplement, pp. II.522-II.523. print.
Meeting Info.: Scientific Sessions 2001 of the American Heart Association. Anaheim, California, USA. November 11-14, 2001. American Heart Association.
CODEN: CIRCAZ. ISSN: 0009-7322.

L6 ANSWER 6 OF 6 BIOSIS COPYRIGHT (c) 2004 The Thomson Corporation. on STN
AU Zhou, Zhong Min [Reprint author]; Marso, Steven P.; Schmidt, Ann Marie; Stern, David M.; Qu, Wu; Forudi, Farhad; Wang, Kai; Lincoff, A. Michael; Topol, Eric J.
TI Blockade of receptor for advanced glycation end-products (RAGE) suppresses neointimal formation in diabetic rat carotid artery injury model.
SO Circulation, (October 31, 2000) Vol. 102, No. 18 Supplement, pp. II.246. print.
Meeting Info.: Abstracts from American Heart Association Scientific Sessions 2000. New Orleans, Louisiana, USA. November 12-15, 2000. American Heart Association.
CODEN: CIRCAZ. ISSN: 0009-7322.

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     (FILE 'HOME' ENTERED AT 17:49:38 ON 19 OCT 2004)

     FILE 'MEDLINE, CAPLUS, BIOSIS, SCISEARCH' ENTERED AT 17:49:56 ON 19 OCT
     2004
L1           216 S RECEPTOR(3A)ADVANCED(W)GLYCATION(W)ENDPRODUCT
L2            85 S SRAGE
L3           283 S L1 OR L2
L4         38783 S RESTENOSIS
L5             9 S L3 AND L4
L6             6 DUP REM L5 (3 DUPLICATES REMOVED)
L7            22 S SOLUBLE(3A)RECEPTOR(3A)ADVANCED(W)GLYCATION(W)ENDPRODUCT
L8            16 DUP REM L7 (6 DUPLICATES REMOVED)

=> d au ti so ab 1-16 l8

L8   ANSWER 1 OF 16  BIOSIS  COPYRIGHT (c) 2004 The Thomson Corporation.  on
     STN
AU   Arancio, Ottavio [Reprint Author]; Battaglia, Fortunato [Reprint Author];
     Lin, Chang; Liu, Shumin [Reprint Author]; Trinchese, Fabrizio [Reprint
     Author]; Chen, Xi; Stern, David; Yan, Shi Du
TI   Administration of soluble RAGE protects spatial memory and synaptic
     function in APP/PS1 mice.
SO   Neurology, (March 11 2003) Vol. 60, No. 5 Supplement 1, pp. A206. print.
     Meeting Info.: 55th Annual Meeting of the American Academy of Neurology.
     Honolulu, Hawaii, USA. March 29-April 05, 2003.
     ISSN: 0028-3878 (ISSN print).

L8   ANSWER 2 OF 16  CAPLUS  COPYRIGHT 2004 ACS on STN
IN   Stern, David M.; Schmidt, Ann-Marie; Marso, Steven; Topol, Eric; Lincoff,
     A. Michael
TI   A method for inhibiting new tissue growth in blood vessels in a patient
     subjected to blood vessel injury
SO   PCT Int. Appl., 43 pp.
     CODEN: PIXXD2
AB   This invention provides for a method for inhibiting new tissue growth in
     blood vessels in a subject, wherein the subject experienced blood vessels
     injury, which comprises administering to the subject a pharmaceutically
     effective amount of an inhibitor of receptor for advanced glycation
     endproduct (RAGE) so as to inhibit new tissue growth in the subject's
     blood vessels. The invention also provides for method for inhibiting
     neointimal formation in blood vessels in a subject, wherein the subject
     experienced blood vessel injury, which comprises administering to the
     subject a pharmaceutically effective amount of an inhibitor of receptor for
     advanced glycation endproduct (RAGE) so as to inhibit neointimal formation
     in the subject's blood vessels. The invention also provides a method for
     preventing exaggerated restenosis in a diabetic subject which comprises
     administering to the subject a pharmaceutically effective amount of an
     inhibitor of receptor for advanced glycation endproduct (RAGE) so as to
     prevent exaggerated restenosis in the subject. In the example provided, a
     significant reduction in neointimal area was observed in fatty Zucker rats
     treated with sol. receptor for advanced
     glycation endproduct following carotid artery injury.

L8   ANSWER 3 OF 16  BIOSIS  COPYRIGHT (c) 2004 The Thomson Corporation.  on
     STN
AU   Schmidt, Ann Marie [Inventor]; Stern, David [Inventor]
TI   Method for inhibiting tumor invasion or spreading in a subject.
SO   Official Gazette of the United States Patent and Trademark Office Patents,
     (Oct. 15, 2002) Vol. 1263, No. 3. http://www.uspto.gov/web/menu/patdata.ht
     ml. e-file.
     CODEN: OGUPE7. ISSN: 0098-1133.
AB   The present invention provides for a method for inhibiting tumor invasion
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or metastasis in a subject which comprises administering to the subject a therapeutically effective amount of a form of **soluble Receptor** for **Advanced Glycation Endproducts** (RAGE). The present invention also provides a method for evaluating the ability of an agent to inhibit tumor invasion in a local cellular environment which comprises: (a) admixing with cell culture media an effective amount of the agent; (b) contacting a tumor cell in cell culture with the media from step (a); (c) determining the amount of spreading of the tumor cell culture, and (d) comparing the amount of spreading of the tumor cell culture determined in step (c) with the amount determined in the absence of the agent, thus evaluating the ability of the agent to inhibit tumor invasion in the local cellular environment. The present invention also provides a pharmaceutical composition which comprises a therapeutically effective amount of the agent evaluated in the aforementioned method and a pharmaceutically acceptable carrier.

L8 ANSWER 4 OF 16 MEDLINE on STN DUPLICATE 1

AU Bonnefont-Rousselot D

TI [Antioxidant and anti-AGE therapeutics: evaluation and perspectives]. Therapeutiques anti-oxydantes et anti-AGE: bilans et perspectives.

SO Journal de la Societe de biologie, (2001) 195 (4) 391-8. Ref: 76 Journal code: 100890617. ISSN: 1295-0661.

AB Diabetic patients exhibit an oxidative stress status, that is an imbalance between reactive oxygen species and antioxidant defences, in favour of the first ones. This oxidative stress, together with formation of advanced glycation endproducts (AGEs), is involved in diabetic complications. It could thus be of great interest to propose antioxidant and/or anti-AGE therapeutics as complementary treatment in these patients. Antioxidants can be classical molecules such as vitamin E, lipoic acid or N-acetylcysteine. Thus, vitamin E supplementation can improve insulin efficiency and glycemic equilibrium, as shown by the decrease of glycaemia, glycated haemoglobin and fructosamine values. In addition, this kind of supplementation lowers plasma lipid peroxidation and oxidizability of low density lipoproteins, which is involved in the atherogenesis process. Moreover, it allows to fight against complications such as retinopathy. A second category is represented by molecules able to fight against the effects of glycation end-products (AGEs). They can act: either by preventing cellular action of AGEs; this is obtained with **soluble receptors** of **advanced glycation endproducts** (sRAGE); or by inhibiting AGE formation (scavenging of reactive carbonyl intermediates). Nucleophilic compounds such as pyridoxamine, tenilsetam, 2,3-diaminophenazone, OPB-9195 or aminoguanidine can act in this way. Aminoguanidine is able to limit the development of the main diabetes-associated complications in animals. A double-blind clinical assay has been conducted in type 2 diabetic patients in the United States and the Canada, in order to determine if aminoguanidine is able to slow down the progression of diabetes-induced nephropathy. We will discuss about another guanidic molecule, i.e. metformin, which is also able to scavenge AGEs, in the last part of this review. A third category of molecules is constituted by oral antidiabetic molecules exhibiting antioxidant properties. They are thiazolidinediones (troglitazone) and sulfonylureas (gliclazide). Troglitazone and gliclazide can thus decrease LDL oxidizability and monocyte adhesion to endothelial cells, which is an early step in the atherogenesis process and which is stimulated by oxidised LDLs. Finally, a prospective way is devoted to oral antidiabetic drugs exhibiting both antioxidant and anti-AGE properties. A very used antidiabetic drug of interest is metformin (dimethylbiguanide), since it can prevent diabetes complications not only by lowering glycaemia, but also by inhibiting AGE formation and by stimulating antioxidant defences. The latter therapeutic approach constitutes a future way in the diabetes area, in order both to obtain a better glycemic control and a least development of diabetic complications.

L8 ANSWER 5 OF 16 BIOSIS COPYRIGHT (c) 2004 The Thomson Corporation. on

STN
AU Bucciarelli, Loredana G. [Reprint author]; Qu, Wu [Reprint author]; Wendt, Thoralf M. [Reprint author]; Goova, Mouza T. [Reprint author]; Bakr, Soliman [Reprint author]; Hwang, Yuying C. [Reprint author]; Stern, David M. [Reprint author]; Schmidt, Ann Marie [Reprint author]; Ramasamy, Ravichandran [Reprint author]
TI Blockade of receptor for AGE (RAGE) suppresses levels of cardiac endothelial- and inducible nitric oxide synthase in diabetic mice.
SO Circulation, (October 31, 2000) Vol. 102, No. 18 Supplement, pp. II.117-II.118. print.
Meeting Info.: Abstracts from American Heart Association Scientific Sessions 2000. New Orleans, Louisiana, USA. November 12-15, 2000. American Heart Association.
CODEN: CIRCAZ. ISSN: 0009-7322.

L8 ANSWER 6 OF 16 CAPLUS COPYRIGHT 2004 ACS on STN
IN Schmidt, Ann Marie; Stern, David
TI Inhibition of tumor invasion or spreading based on a **soluble receptor** for **advanced glycation endproducts**
SO PCT Int. Appl., 88 pp.
CODEN: PIXXD2
AB The present invention provides for a method for inhibiting tumor invasion or metastasis in a subject which comprises administering to the subject a therapeutically effective amount of a form of **sol. receptor** for **advanced glycation endproducts** (RAGE). Interruption of cellular RAGE-extracellular matrix (amphoterin and/or similar structures) interaction appears to be at least one mechanism by which sRAGE limits tumor growth. The present invention also provides a method for evaluating the ability of an agent to inhibit tumor invasion in a local cellular environment which comprises: (a) admixing with cell culture media an effective amount of the agent; (b) contacting a tumor cell in cell culture with the media from step (a); (c) determining the amount of spreading of the tumor cell culture, and (d) comparing the amount of spreading of the tumor cell culture determined in step (c) with the amount determined in the absence of the agent, thus evaluating the ability of the agent to inhibit tumor invasion in the local cellular environment. The present invention also provides a pharmaceutical composition which comprises a therapeutically effective amount of the agent evaluated in the aforementioned method and a pharmaceutically acceptable carrier.

L8 ANSWER 7 OF 16 CAPLUS COPYRIGHT 2004 ACS on STN
IN Stern, David; Schmidt, Ann Marie
TI Method to prevent accelerated atherosclerosis using **soluble receptor** for **advanced glycation endproducts** (sRAGE)
SO PCT Int. Appl., 53 pp.
CODEN: PIXXD2
AB A method is provided for prevention of accelerated atherosclerosis in a subject predisposed thereto which comprises administering to the subject a polypeptide derived from **sol. receptor** for **advanced glycation endproduct** in an amount effective to prevent accelerated atherosclerosis in the subject. Also provided is a method to prevent a macrovessel disease in a subject predisposed thereto which comprises administering to the subject a polypeptide derived from **sol. receptor** for **advanced glycation endproduct** in an amount effective to prevent macrovessel disease in the subject.

L8 ANSWER 8 OF 16 SCISEARCH COPYRIGHT (c) 2004 The Thomson Corporation. on STN

AU Li J (Reprint); Wu J; Stern D M; Schmidt A M
TI Administration of **soluble Receptor** for **Advanced Glycation Endproducts** (sRAGE) enhances wound repair in diabetic mice.
SO CIRCULATION, (2 NOV 1999) Vol. 100, No. 18, Supp. [S], pp. 3651-3651. Publisher: LIPPINCOTT WILLIAMS & WILKINS, 530 WALNUT ST, PHILADELPHIA, PA 19106-3621.
ISSN: 0009-7322.

L8 ANSWER 9 OF 16 BIOSIS COPYRIGHT (c) 2004 The Thomson Corporation. on STN DUPLICATE 2
AU Salahudeen, A. K. [Reprint author]; Huang, H. [Reprint author]; Stern, D.; Schmidt, A. M.
TI Administration of **soluble receptor** for **advanced glycation endproducts** (sRAGE) in DB-DB mice suppresses abnormalities in the early and late stages of diabetic nephropathy.
SO FASEB Journal, (March 12, 1999) Vol. 13, No. 4 PART 1, pp. A216. print.
Meeting Info.: Annual Meeting of the Professional Research Scientists for Experimental Biology 99. Washington, D.C., USA. April 17-21, 1999.
CODEN: FAJOEC. ISSN: 0892-6638.

L8 ANSWER 10 OF 16 BIOSIS COPYRIGHT (c) 2004 The Thomson Corporation. on STN DUPLICATE 3
AU Salahudeen, A. K. [Reprint author]; Huang, H. [Reprint author]; Stern, D.; Schmidt, A. M.
TI Administration of **soluble receptor** for **advanced glycation endproducts** (sRAGE) in DB-DB mice suppresses abnormalities in the early and late stages of diabetic nephropathy.
SO Journal of Investigative Medicine, (April, 1999) Vol. 47, No. 4, pp. 207A. print.
Meeting Info.: Meeting of the American Federation For Medical Research at Experimental Biology '99. Washington, D.C., USA. April 16-18, 1999. American Federation for Medical Research.
ISSN: 1081-5589.

L8 ANSWER 11 OF 16 BIOSIS COPYRIGHT (c) 2004 The Thomson Corporation. on STN
AU Li, Jun [Reprint author]; Wu, June [Reprint author]; Stern, David M. [Reprint author]; Schmidt, Ann Marie [Reprint author]
TI Administration of **soluble receptor** for **advanced glycation endproducts** (sRAGE) enhances wound repair in diabetic mice.
SO Circulation, (Nov. 2, 1999) Vol. 100, No. 18 SUPPL., pp. I.692. print.
Meeting Info.: 72nd Scientific Sessions of the American Heart Association. Atlanta, Georgia, USA. November 7-10, 1999.
CODEN: CIRCAZ. ISSN: 0009-7322.

L8 ANSWER 12 OF 16 CAPLUS COPYRIGHT 2004 ACS on STN
IN Stern, David M.; Schmidt, Ann Marie
TI Method for treating symptoms of diabetes with agents preventing binding of advanced glycation endproducts to receptors
SO PCT Int. Appl., 33 pp.
CODEN: PIXXD2
AB A method is provided for treating symptoms of diabetes in a diabetic subject, e.g. abnormal wound healing, which comprises administering to the subject a therapeutically effective amount of an agent which inhibits binding of advanced glycation endproducts to any receptor for advanced glycation endproducts so as to treat chronic symptoms of diabetes in the subject. Improved wound healing in diabetic mice by treatment with the **sol. receptor** for **advanced glycation endproducts** is described.

L8 ANSWER 13 OF 16 MEDLINE on STN DUPLICATE 4
AU Park L; Raman K G; Lee K J; Lu Y; Ferran L J Jr; Chow W S; Stern D; Schmidt A M
TI Suppression of accelerated diabetic atherosclerosis by the **soluble receptor** for **advanced glycation endproducts**.
SO Nature medicine, (1998 Sep) 4 (9) 1025-31.
Journal code: 9502015. ISSN: 1078-8956.
AB Accelerated atherosclerosis in patients with diabetes is a major cause of their morbidity and mortality, and it is unresponsive to therapy aimed at restoring relative euglycemia. In hyperglycemia, nonenzymatic glycation and oxidation of proteins and lipids results in the accumulation of irreversibly formed advanced glycation endproducts. These advanced glycation endproducts engage their receptor in cells of the blood vessel wall, thereby activating mechanisms linked to the development of vascular lesions. We report here a model of accelerated and advanced atherosclerosis in diabetic mice deficient for apolipoprotein E. Treatment of these mice with the soluble extracellular domain of the receptor for advanced glycation endproducts completely suppressed diabetic atherosclerosis in a glycemia- and lipid-independent manner. These findings indicate interaction between the advanced glycation endproducts and their receptor is involved in the development of accelerated atherosclerosis in diabetes, and identify this receptor as a new therapeutic target in diabetic macrovascular disease.

L8 ANSWER 14 OF 16 BIOSIS COPYRIGHT (c) 2004 The Thomson Corporation. on STN
AU Makker, Gotam [Reprint author]; Vorp, David A.; Lindenberg, Noah; Fan, Linda; Wang, David H.-J.; Qu, Wu; Stern, David M.; Schmidt, Ann Marie [Reprint author]
TI Maintenance of vascular structural integrity in diabetic LDL receptor null mice treated with soluble receptor for AGE (sRAGE).
SO Circulation, (Oct. 27, 1998) Vol. 98, No. 17 SUPPL., pp. I12. print.
Meeting Info.: 71st Scientific Sessions of the American Heart Association. Dallas, Texas, USA. November 8-11, 1998. The American Heart Association.
CODEN: CIRCAZ. ISSN: 0009-7322.

L8 ANSWER 15 OF 16 SCISEARCH COPYRIGHT (c) 2004 The Thomson Corporation. on STN
AU Park L (Reprint); Raman K G; Lee K J; Lu Y; Ginsberg M D; Ferran L; Stern D M; Schmidt A M
TI A murine model of accelerated diabetic atherosclerosis: Suppression by **soluble receptor** for **advanced glycation endproducts**
SO CIRCULATION, (21 OCT 1997) Vol. 96, No. 8, Supp. [S], pp. 3079-3079.
Publisher: AMER HEART ASSOC, 7272 GREENVILLE AVENUE, DALLAS, TX 75231-4596.
ISSN: 0009-7322.

L8 ANSWER 16 OF 16 BIOSIS COPYRIGHT (c) 2004 The Thomson Corporation. on STN
AU Wautier, J. L. [Reprint author]; Zoukourian, C.; Chappey, O.; Wautier, M. P.; Guillauseau, P. J.; Cao, R.; Hori, O.; Stern, D.; Schmidt, A. M.
TI Receptor-mediated endothelial dysfunction in diabetic vasculopathy: **Soluble receptor** for **advanced glycation endproducts** blocks hyperpermeability.
SO Journal of Investigative Medicine, (1995) Vol. 43, No. SUPPL. 2, pp. 215A.
Meeting Info.: Clinical Research Meeting. San Diego, California, USA. May 5-8, 1995.

=> d pi 6 7 12 18

L8 ANSWER 6 OF 16 CAPLUS COPYRIGHT 2004 ACS on STN

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 9954485	A1	19991028	WO 1999-US8427	19990416
	W: AE, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
	RW: GH, GM, KE, LS, MW, SD, SL, SZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
	US 6465422	B1	20021015	US 1998-62365	19980417
	CA 2325573	AA	19991028	CA 1999-2325573	19990416
	AU 9934957	A1	19991108	AU 1999-34957	19990416
	EP 1071794	A1	20010131	EP 1999-916699	19990416
	R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI				
	JP 2002512038	T2	20020423	JP 2000-544814	19990416
	US 2002177550	A1	20021128	US 2001-851071	20010508

L8 ANSWER 7 OF 16 CAPLUS COPYRIGHT 2004 ACS on STN

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 9907402	A1	19990218	WO 1998-US16303	19980805
	W: AU, CA, JP, MX, US				
	RW: AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE				
	US 2001039256	A1	20011108	US 1997-905709	19970805
	AU 9888239	A1	19990301	AU 1998-88239	19980805
	AU 758252	B2	20030320		
	EP 1011706	A1	20000628	EP 1998-939876	19980805
	EP 1011706	B1	20031112		
	R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI				
	JP 2001513511	T2	20010904	JP 2000-506991	19980805
	AT 253929	E	20031115	AT 1998-939876	19980805
	PT 1011706	T	20040430	PT 1998-939876	19980805

L8 ANSWER 12 OF 16 CAPLUS COPYRIGHT 2004 ACS on STN

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 9822138	A1	19980528	WO 1997-US21197	19971112
	W: AU, CA, JP, MX				
	RW: AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE				
	US 2003059423	A1	20030327	US 1996-755235	19961122
	US 6790443	B2	20040914		
	CA 2271857	AA	19980528	CA 1997-2271857	19971112
	AU 9852639	A1	19980610	AU 1998-52639	19971112
	AU 745241	B2	20020314		
	EP 946196	A1	19991006	EP 1997-947592	19971112
	R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI				
	JP 2001504493	T2	20010403	JP 1998-523860	19971112

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